ENCE of hypergloallinemia and disproteinemia (New Eng J Med 281:1428, 1969). Our simplified and practical approach to the diagnosis, surveillance, and treatment of the whole spectrum of cases of consumption coagulopathy has been described elsewhere.† No single test is universally useful in the diagnosis and management of consumption coagulopathy. We have not yet uncovered all the secrets that the test may yield to us. In our view this test of Brebn and his deservesthe most serious attention from those interested in problems of consumption coagulopathy.

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Nalbandian RM, Henry RL, Camp FR Jr: A Practical Synopsis of Consumption Coagulopathy (United States Army Medical Research Laboratory Report No 8922). Fort Knox, Kentucky, United States Army Medical Research and Development Command, August 28, 1970

Nalbandian RM, Henry RL, Kessler DL: Consumption Coagulopathy: Practical principles of diagnosis and management (United States Army Medical Research Laboratory Report No 912). Fort Knox, Kentucky, United States Army Medical Research and Development Command, November 30, 1970

IDIOPATHIC HYPERCALCIURIA

To the Editor: The paper by Finn et al. in the December 31, 1970, issue of the Journal, entitled "Transplantation of a Kidney from a Patient with Idiopathic Hypercalcemia," purports to show that idiopathic hypercalcemia is caused by excessive gastrointestinal absorption of calcium rather than by an inability of the kidney to conserve calcium. The kidney of a donor with idiopathic hypercalcemia was transplanted into a recipient who did not go on to acquire idiopathic hypercalcemia himself. The donor, however, persisted in having this abnormality.

The problem with the conclusion reached from this retrospective case is that the recipient was, of course, placed on steroids after the transplant, and steroids are known to decrease intestinal absorption of calcium, as indicated by Harrison and Harrison, in their article entitled "Transfer of Ca across Intestinal Wall in Vitro in Relation to Action of Vitamin D and Cortisol." (Amer J Physiol 199:285, 1960). This could have affected his urinary excretion of calcium also.

If one wanted to use this case to demonstrate the primary role of increased gastrointestinal absorption of calcium in idiopathic hypercalcuria, one would have had to place the donor with idiopathic hypercalcuria on steroids, or a dietary calcium of only 150 mg per day, before nephrectomy and transplantation. If the donor had responded to this program by decreasing his urinary calcium excretion to normal levels before transplantation, the post-transplant data presented in their paper would have been unnecessary. However, if the donor's urinary calcium excretion had continued to be high under these circumstances, the recipient's normal urinary calcium excretion after transplantation would have been important, indicating a nonrenal origin for the condition.

If this study had been carried out before transplantation, we would have more meaningful data. I agree with the authors' basic contention, but believe that they missed a chance to prove it. Transplantation can only help us with this question if we take a patient as donor whose calcium excretion in the urine does not decrease to normal despite the severest calcium restriction in the diet, or if steroids are not given after operation to the recipient.

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The above letter was referred to the authors of the article in question, who offer the following reply:

To the Editor: The study was not a retrospective one, and calcium balance studies carried out in the donor before nephrectomy demonstrated hypercalcuria on a diet of both 300 mg and 1 g of calcium. Hypercalcuria disappeared immediately in the transplanted kidney, but balance studies were carried out one year later to avoid the effect that chronic uremia might have had on calcium metabolism in the early post-transplantation period. Contrary to Dr. Silber's opinion, studies of patients with idiopathic hypercalcemia have demonstrated that although urinary calcium decreases with calcium restriction, negative calcium balance occurs with inappropriately high urinary calcium for 10 to 18 days. Whether negative calcium balance would persist during a longer period of severe restriction of calcium intake is unknown. Reducing calcium intake for a brief period, however, does not differentiate these populations of patients, one with a primary renal leak of calcium and another with excessive gastrointestinal absorption.

Regarding Dr. Silber's objection that steroids were not given to the donor before nephrectomy, as we pointed out in our article, all studies of idiopathic hypercalcemia in which steroids have been administered demonstrate further increase in urinary excretion, and we did not consider it justifiable to subject the donor to this potential risk.

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